REMARKS

Favorable reconsideration is respectfully requested in view of the following remarks and the remarks of record.

Claim 14 is pending and rejected.

On pages 2-13, claim 14 was rejected under 35 U.S.C. § 103(a) as being unpatentable over Nordisk (WO 98/58646) in view of Perez-Santonja et al. (Am J. Ophthalmol., 1999, 127: 497-504), WO 98/44922 (Yang et al.) and WO 97/43278 (Ankersen et al.) as evidenced by Suzuki et al. (see p. 550, abstract, Suzuki et al. Curr. Eye Res., 2000, 21: 550-553) and Fini et al. (see p. 812 2nd col., Arch Dermatol. Res., 1998, 290: 812-823) and the data of cornea (p.3-4, retrieved from the **NEI** website, www.nei.nih.gov/health/cornealdisease).

Applicants respectfully traverse this rejection for the following reasons.

Nordisk describes that Somatostatin receptors (SSTR 4 and SSTR 2) are expressed in the iris-ciliary body and retina, and refers to treating glaucoma, stroma keratitis, iritis, retinitis, cataract and conjunctivitis. However, Suzuki and Fini do not describe that the diseases which Nordisk discloses result in decreased corneal sensitivity. The other cited references fail to remedy this deficiency. Thus, Nordisk either alone or in combination fails to teach or suggest the claimed method.

Furthermore, the present specification indicates in Experimental Example 4 the effect of octreotide, a somatostatin analog, on axon extension of cultured rabbit trigeminal nerve cells. In Experimental Example 4, when calculating the percentage of the cells including an axon having a length of not less than twice the diameter of the cell by comparing Octreotide (10μM) and a control, Octreotide (10μM) was shown to promote axon extension.

Furthermore, the present specification indicates in Experimental Example 5 the effect of the claimed compounds, SSTR2 specific agonist (Compound 1) and SSTR4 specific agonist (Compound 2) on axon extension of cultured rabbit trigeminal nerve cells. In Experimental Example 5, increases in neuritogenic cells were examined by measuring the absorbance to indicate neurofilament amount. Compound 1 and Compound 2 promoted axon extension at 1μM and 0.1μM, respectively, as compared to a control. Thus, Octreotide promotes axon extension at 10μM, while the claimed compounds promote axon extension at 1μM and 0.1μM. Hence, the claimed compounds exert a promoting effect on axon extension at a 10 or 100 times lower concentration as compared to Octreotide.

One of ordinary skill in the art would not expect the potency of the claimed invention, from Nordisk in view of Perez-Santoja, Yang and Ankersen as evidenced by Suzuki and Fini and the data of cornea (NEI website). Accordingly, the claimed invention is not obvious over the combination of the above references. Thus, this rejection is untenable and should be withdrawn.

In view of the foregoing amendments and remarks, it is respectfully submitted that the present application is in condition for allowance and early notice to that effect is hereby requested.

If the Examiner has any comments or proposals for expediting prosecution, please contact the undersigned attorney at the telephone number below.

Respectfully submitted,

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By /William R. Schmidt, II/

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